Reports
Lewis,
R.

LIVE OAK DECLINE IN TEXAS

VTLS#252469
File
Author File

FILE

1979

FINAL REPORT

LIVE OAK DECLINE IN TEXAS

Robert Lewis, Jr.
Research Plant Pathologist

USDA Forest Service
Southern Hardwoods Laboratory
Stoneville, Mississippi

Southern Forest Experiment Station

FINAL REPORT

LIVE OAK DECLINE IN TEXAS

Abstract

Cephalosporium diospyri is not the primary cause of wilt or "decline" in Texas live oaks as was previously believed. The fungus can be isolated from healthy live oaks throughout the South where "decline" is not a problem and healthy live oaks inoculated with it do not develop wilt. The primary cause is actually Ceratocystis fagacearum (oak wilt) and the disease should be correctly called oak wilt instead of "oak decline". Oak wilt in Texas is stimulated by mild or cool spring and fall temperatures and is suppressed by hot summer temperatures. Live oak root temperatures are much cooler than ambient temperatures during summer and C. fagacearum over-summers in them. Live oaks can be killed rapidly or slowly by the wilt alone but sometimes wilt-affected trees are colonized by canker fungi, i.e., Botryodiplodia theobromae which cause additional dieback. Oak wilt is spread through root grafts and by unidentified insect vectors. The insect vectors are believed to be responsible for starting new infection centers. The wide distribution and severity of oak wilt in Texas indicate that it has been in the State for many years. It is suggested that oak wilt might have been in Texas even before it was identified in Wisconsin for the first time anywhere. Theories suggesting that oak wilt cannot be a serious disease in the South due to lethal high summer temperatures are not true. Oak wilt can be suppressed with systemic fungicides (Arbotect and Lignasan) if the treatments are applied during initial phases of infection. Summer applications appear to be more effective than spring and fall treatments.

This might be due to an attenuation of the fungus by high summer temperatures. Wilt preventative treatments are also effective.

FINAL REPORT

LIVE OAK DECLINE IN TEXAS

Robert Lewis, Jr.

Research Plant Pathologist, U. S. Forest Service, Southern Forest Experiment Station, Southern Hardwoods Laboratory, Stoneville, Mississippi.

Background

Live oak is the primary hardwood species in much of Central Texas. It has high aesthetic and historic values. It also provides food and shelter for wildlife; helps protect sites from soil erosion; provides a relatively cool environment and shade for man and livestock; and provides a renewable source of home-heating fuel.

Rural and urban communities, parks and resort areas have been developed and expanded around live oaks with a hope of enduring beauty and shade. However, this hope has been diminished by a disease previously known as "live oak decline". The disease has seriously affected much of the Texas "Hill Country" and left some hills, slopes, and valleys almost barren.

The disease was first reported in Austin, Texas, by Taubenhaus (1934, 1935). It was described as a serious malady that could kill live oaks rapidly but the causal agent was not identified.

Dunlap and Harrison (1949) studied the disease for 9 years and described the symptoms as leaf yellowing or mottling, defoliation, dieback, resprouting from roots, and death of trees. They observed that

when symptoms developed over the entire crown, death usually occurred within a few weeks but when symptoms developed in one branch at a time, one or two seasons elapsed before trees were killed. The disease occurred in young and old trees on acid, highly alkaline, sandy, heavy clay, dry and wet soils in both hills and valleys. The cause of the disease was not determined but a biological agent, perhaps a virus, was suspected.

Halliwell (1964, 1965, 1966) studied the disease during the 1960's but did not find a virus associated with it. His description of symptoms was similar to those given by Dunlap and Harrison (1949). He also reported a fast decline resembling oak wilt and a slow decline. Cephalosporium was believed to cause slow decline and Hyalodendron sp. was suspected in the fast decline.

Van Arsdel has been studying the disease since 1968. He identified it as a vascular wilt caused by Cephalosporium diospyri (1970) and called it "oak decline". Cephalosporium diospyri was believed to be a highly evolved parasite that requires 10 or more years to kill live oaks (Van Arsdel et al. 1974). However, other published reports described a disease that often killed live oaks within a few weeks instead of 10 years (Taubenhaus 1935, Dunlap and Harrison 1949, and Halliwell 1965).

While confusion and conflicting reports about the etiology and control of "live oak decline" surfaced in the mid 1970's; the disease continued to spread freely across much of Texas and there was little hope for saving affected trees.

The USDI National Park Service, in cooperation with the U. S. Forest Service, Southern Forest Experiment Station, sponsored research in 1976 to explain the causes of "live oak decline" in Texas and to develop some methods of control. This is the final report of the 3-year study.

Etiology of the Live Oak Disease

An Attempt to Associate the Disease With Insect Defoliation

The initial phase of research was to determine if insect defoliation could be associated with decline of live oaks. One theory was that trees might be weakened by repeated insect defoliations and then slowly killed by *Cephalosporium diospyri*.

Several species of defoliating insects were observed in Central Texas during 1976-1979 but substantial defoliation did not occur until spring 1979. Trees that were defoliated in 1979 by the spring canker worm have refoliated. Some of the new leaves appeared reddish and others were somewhat chloritic in June but most were back to normal colors by late summer. The incidence of disease outbreaks did not appear to be affected by the 1979 defoliation nor by insect defoliation in the previous 3 years. The association of disease with insect defoliation could not be made after 3 years.

Analysis of Cephalosporium diospyri as a Potential Cause of Decline

Cephalosporium diospyri is the first fungus that we considered as a possible cause of decline. It had been reported by other researchers as the primary pathogen for about 10 years. The objective of this phase of study was to associate the fungus with diseased trees in the field and demonstrate its ability to produce similar disease in artificially inoculated trees.

Samples were taken from live oaks with advanced "decline" symptoms (50% or more crown dieback) in August 1976 and assayed for Cephalosporium diospyri. Twigs, roots, and increment cores were taken from trees and attempts were made to isolate C. diospyri. Bark-free tissue from each tree was surface-disinfested in a 0.2% sodium hypochlorite solution for 0.5-1.5 min. and then placed on potato dextrose agar (PDA) and 0.8% wood chip agar. Fungi grew from plant tissues and were identified within 3 weeks. Cephalosporium diospyri was isolated from 4 of 15 living trees and from 2 dead trees. Other fungi including some potential pathogens were also isolated. These included Alternaria sp., Aspergillus spp., Botryodiplodia theobromae, Coryneum sp., Cytospora sp., Endothia sp., Epicoccum sp., Fusarium sp., Gloeosporium sp., Nigrospora sp., Penicillium spp., Pestalotia sp., Rabenhorstia sp., Sporidesmium sp., and Trichoderma sp. Some of these fungi were isolated more frequently than C. diospyri.

Inoculations were made with various C. diospyri isolates during 1977-1979 to see if the fungus could cause wilting. Two- and 3-year-old pot planted live oaks were inoculated with C. diospyri spores mixed in sterile water. A drop of spore mixture was placed on a surafce-disinfested area of the lower stem and two incisions made through the liquid into xylem with a sterile razor blade allowing spores to be sucked into vessels. Wilt or decline symptoms did not develop in any of 65 trees inoculated at 28-32^{0} C but C. diospyri was consistently re-isolated (Table 1). Cephalosporium diospyri grew in healthy live oaks but did not cause wilting.

Table 1.--Inoculation of healthy live oaks with _Cephalosporium diospyri_ at 28-32° C.

Date inoculated	Trees inoculated (No.)	Observation time (Months)	Wilt development (No.)
February 1977	6	7	0
March 1977	6	6	0
April 1977	4	6	0
May 1977	4	6	0
June 1977	10	6	0
August 1977	4	6	0
June 1978	6	6	0
February 1979	9	7	0
March 1979	3	6	0
July 1979	3	3	0
September 1979	10	1.5	0

Relatively healthy oaks outside of Central Texas were assayed to see if _C. diospyri_ grew in them. Twig samples were taken and fungi were isolated and identified on PDA. _Cephalosporium diospyri_ was isolated from 18 oaks that were not showing typical "decline" symptoms (Table 2). Two of these 18 had been partially defoliated by insects in Florida but did not have any other symptoms. Relatively healthy oaks were colonized by _C. diospyri_ over a wide geographical area.

Cephalosporium diospyri did not cause live oak wilting and was found in relatively healthy trees at various locations. It did not qualify as the primary cause of live oak wilt or decline.

Discovery of a Primary Pathogen

The search for a primary pathogen was intensified in 1977. It was cool and wet in Central Texas during April and wilt was not expected since we were still considering _C. diospyri_ as the primary pathogen and it is favored by high temperatures (Van Arsdel and Bush 1970) and relatively dry conditions. However, live oaks were found with active wilt at the Kerrville State Park. Leaf symptoms were exactly like those described by Halliwell (1966) for what he believed to be _Cephalosporium_ wilt. The symptoms were leaf desiccation and necrosis from the margins, chlorotic veins and leaf mottling (Fig. 1). Two live oaks at the LBJ Ranch, and two at the Kerrville State Park in addition to one Spanish red oak with symptoms were assayed for wilt fungi in April.

Figure 1.--Kerrville, Texas, live oak with initial wilt in April 1977. Note desiccation of leaf margins (whitish edges), mottling (yellow leaf on left), vein chlorosis, and small leaves (on twigs with flowers).

Table 2.--Isolation of Cephalosporium diospyri from oaks without typical decline symptoms.

Location	Species	Date collected	No. trees
Oxford, MS	Post oak	May 1978	1
Stoneville, MS (Delta Exp. For.)	Water oak	June 1978	1
Gulfport, MS	Live oak	August 1978	5
Tyler, TX	Live oak	September 1978	3
Webb, MS	Live oak	May 1979	1
De Soto Park, FL	Live oak	May 1979	1
Hernando, FL	Live oak	May 1979	1
Ocala, FL	Laurel oak	May 1979	2
Tampa, FL	Laurel oak	May 1979	3

A fungus with brown conidiophores resembling vegetative hyphae and producing endogenous, hyaline, cylindrical, truncate conidia was isolated from all 5 trees with incipient wilt (Fig. 2). It was identified as Endoconidiophora sp., an asexual stage in the life cycles of some fungi in the genus Ceratocystis. A fruity odor, characteristic of Ceratocystis fagacearum (oak wilt), was produced by the fungous cultures.

The five isolates of Endoconidiophora sp. were compared and mated with Ceratocystis fagacearum strains obtained from the American Type Culture Collection (ATCC). Strains "A" (ATCC #24789) and "B" (ATCC #24790) were used. The asexual ATCC strains were taxonomically identical to the five Texas isolates. When the Texas isolates were mated with strain "A" or "B" (ATCC), perithecia and ascospores of C. fagacearum were produced. The Texas isolates were typed by their response to ATCC strains. Texas strain "A" produced perithecia when mated with ATCC strain "B". Texas strain "B" successfully mated with ATCC strain "A". When Texas strains "A" and "B" were mixed in cultures, perithecia and ascospores of C. fagacearum were produced. Oak wilt was positively identified in the 5 trees with incipient wilt upon completion of cultural and taxonomic studies of isolated fungi.

Preliminary inoculation experiments were conducted in May 1977 to test the pathogenicity of C. fagacearum from live oaks in Texas. Six healthy live oaks, 2 Nuttall and 2 cherrybark oaks were inoculated with conidia at 26^{o} C. The trees developed wilt similar to that observed in the field within 10-19 days after inoculation and were killed to the root collar after 6 weeks. Some trees resprouted from the root collar as had been reported for "declining" live oaks (Fig. 3).

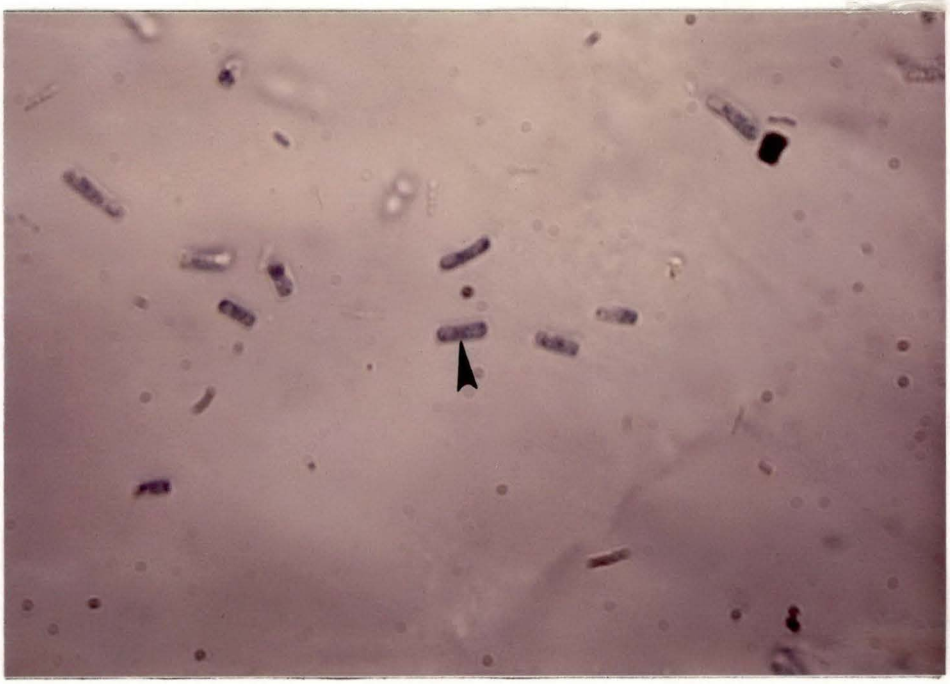

Figure 2.--Conidia of Ceratocystis fagacearum isolated from Kerrville, Texas, live oak in April 1977. Conidia are stained blue.

Figure 3.--Live oak killed to the root collar by Ceratocystis fagacearum 6 weeks after inoculation with conidia in May 1977. Note the characteristic resprouting (green leaf) from the root collar.

Ceratocystis fagacearum was reisolated from the root collars or roots of inoculated trees.

Discovery of C. fagacearum in Central Texas during spring 1977 and demonstration of its virulence in healthy live oaks were key clues used in subsequent research to resolve the "oak decline" question. The fungus has been known as a highly virulent primary pathogen of most oaks in northerly states for many years but was not considered a serious threat to oaks in the South due to high summer temperatures. Ceratocystis fagacearum was identified as a primary pathogen of Texas live oaks but not necessarily the primary cause of "decline" at this stage of the research.

Oak Wilt Identified as the Primary Disease of Texas Live Oaks

Study plots were established in 1977 and 1978 to allow systematic and detailed observations of live oak disease developments. New and old infection centers were included in the plots located in the vicinity of Kerrville, Texas.

Healthy, wilting, and previously wilted live oaks were assayed for pathogenic fungi during spring, summer and fall. Healthy trees were in infection centers or near them but were not grafted to diseased trees; wilting trees were in the initial and most active phase of the pathologic development and showed no signs of previous wilt; previously wilted live oaks had been affected by wilt for 3 or more months. Two twig samples were taken from each tree and chilled in an ice chest until stored in a refrigerator. Fungi were isolated and identified from each sample.

Ceratocystis fagacearum was isolated from most live oaks with initial wilt but was not isolated from many of the same trees 3 or more months later. It was isolated mainly during spring and fall but was occasionally isolated during summer (Table 3).

Cephalosporium spp. and *Phialophora* spp. were also isolated from some of the sampled trees. Since both fungi are morphologically similar, they were placed in one category. These fungi were isolated during all seasons and from healthy as well as diseased trees (Table 4).

Samples from 361 trees in 32 separate infection centers were analyzed during 1977-1979. *Ceratocystis fagacearum* was isolated from 81% of the trees with initial wilt and 12% of the trees which had been previously wilted but was not isolated from healthy trees. *Cephalosporium* and *Phialophora* were frequently isolated from healthy and previously wilted trees but *C. fagacearum* was isolated mainly from trees with initial wilt (Table 5).

Ceratocystis fagacearum was consistently associated with wilt in Texas oaks and caused wilt in all inoculated healthy trees. Evidence of other fungi capable of causing the wilt was not obtained. It is concluded that the primary lethal disease of Texas live oaks is oak wilt caused by *C. fagacearum*.

Diagnosing Oak Wilt in Texas

Oak wilt might be more difficult to identify in Texas than in northerly states due to variations in symptoms. The symptoms in Texas are generally the same as those described by Dunlap and Harrison (1949) and Halliwell (1964) for "oak decline".

Table 3.--Isolation of Ceratocystis fagacearum from twigs of live oaks at various stages of wilt in Central Texas during spring, summer and fall.

Date Year	Date Season	Symptom stage	No. trees	Pct with C. fagacearum
1977	Spring	Initial wilting	13	92
		Advanced decline	74	0
	Summer	Advanced decline	15	0
	Fall	Healthy	13	0
		Initial wilt	55	67
		Wilted during spring	25	24
1978	Spring	Healthy	11	0
		Initial wilting	24	88
		Wilted during fall 1977	30	43
	Summer	Healthy	18	0
		Wilting Spanish red oak	3	100
		Wilted during spring	28	7
	Fall	Initial wilting	13	92
1979	Spring	Healthy	13	0
		Initial wilting	24	96
		Symptomless but grafted to wilting	3	100
		Wilted during fall 1978	5	0

Table 4.--Isolation of *Cephalosporium* and *Phialophora* spp. from twigs of live oaks at various stages of wilt in Central Texas during spring, summer and fall.

Date: Year	Date: Season	Symptom stage	No. trees	Pct with *Cephalosporium* or *Phialophora*
1977	Spring	Initial wilting	13	23
		Advanced decline	74	55
	Summer	Advanced decline	15	47
	Fall	Healthy	13	31
		Initial wilting	55	18
		Wilted during spring	25	68
1978	Spring	Healthy	11	73
		Initial wilting	24	13
		Wilted during fall 1977	30	47
	Summer	Healthy	18	33
		Wilted during spring	28	14
	Fall	Initial wilt	13	15
1979	Spring	Healthy	13	61
		Initial wilt	24	21
		Wilted during fall 1978	5	40
	Summer			

Table 5.--Isolation of *Ceratocystis fagacearum*, *Cephalosporium* spp. and *Phialophora* spp. from live oaks during 1977-1979.

Symptom stage	No. trees	*C. fagacearum*	*Cephalosporium* and *Phialophora*
		----------Pct recovery	------------
Healthy	55	0	47
Initial wilting	129	81	18
Previously wilted	177	12	48

Symptoms were studied during initial oak wilt development in 1977-1979. Leaf chlorosis and mottling followed by moderate to heavy defoliation were among the first symptoms observed (Fig. 4). The symptoms were on all or parts of each tree. Other symptoms were marginal leaf necrosis and vein chlorosis (Fig. 1) often followed by defoliation. Sometimes trees die very rapidly and most of the leaves remain attached. The tree in figure 4 died within 2 months after the photograph was made and most of the chlorotic leaves had turned brown and about 50% of them remained attached.

Symptoms were studied from time of infection until trees died. Advanced symptoms developed within 3 months of infection and were small and often chlorotic leaves, dieback, and resprouting from main stems and larger limbs (Figs. 5 and 6). Roots of most affected trees remained alive for several years after wilt was initiated but crowns were 50-100% dead after 1 year.

When limbs and twigs survived oak wilt for 3 or more months, subsequent symptom development was slow. The most noticeable development was a continuation of dieback. Even though later stages sometimes consisted of slow dieback, the ultimate effect of oak wilt was death of infected trees (Fig. 7).

Symptoms can be used to help diagnose oak wilt in Texas but they may vary depending on the climate and amount of infection. Positive identification can be made by culturing the fungus from infected trees. All symptoms described and illustrated in this section have been confirmed as oak wilt by culturing C. fagacearum from infected trees and reproducing similar disease symptoms in artificially inoculated live oaks.

Figure 4.--Live oak with initial wilt symptoms throughout crown. Notice chlorotic leaves (insert) and brown leaves on lower crown near center and compare with green foliage on healthy live oaks in background. Also notice the full crown of this tree during initial phase of wilt.

Figure 5.--Live oak with initial phase of oak wilt in November 1977. Only part of the crown is showing chlorotic and brown leaves and defoliation. The same tree is shown with advanced symptoms in figure 6.

Figure 6.--Live oak with advanced phase of oak wilt in April 1978. Top photograph, notice the small chlorotic leaves, thin crown, dieback and resprouting from the bole. The entire crown was dead in July 1978. Bottom photograph, compare with Figure 5 showing initial wilt in November 1977.

Figure 7.--Large live oaks in Kerrville, Texas, killed by oak wilt in 1978.

Temperature Affects C. fagacearum Survival and Wilt Development in Texas

The effect of high temperatures on *C. fagacearum* growth was tested. Three isolates of the fungus were placed on PDA and in a growth chamber at 31-32^{o} C. Growth did not occur after 10 days. However, the 3 isolates grew well at 26^{o} C. Texas isolates of *C. fagacearum* were sensitive to high temperatures.

The effect of moderate and high temperatures on wilt development was studied. Healthy live oaks were inoculated with *C. fagacearum* (Texas) spores and placed in growth chambers at 26^{o} C and 31^{o} C. Wilt developed in all trees inoculated at 26^{o} C but did not develop in trees inoculated at 31^{o} C (Table 6). Oak wilt from Texas isolates of *C. fagacearum* was favored by a moderate temperature and inhibited by a high temperature.

Weather data were studied and related to seasonal wilt development. Records of mean monthly temperatures in Kerrville, Texas (1941-1970) were obtained from the National Weather Service. These data are summarized in Table 7. July and August were the hottest months. These are also the months when initial oak wilt symptoms were seldom observed. Symptoms were initiated mainly during April - June and September - November. Temperatures were much cooler and suitable for oak wilt during these months than in July and August. Daytime temperatures often approached 38^{o} C (100^{o} F) in July and August. Oak wilt was affected by seasonal differences in temperatures.

The effects of below normal seasonal temperatures on oak wilt development were observed in 1979. Below normal temperatures in Texas were reported by the National Weather Service for parts of 1979.

Table 6.--Wilt development in live oaks inoculated with _Ceratocystis fagacearum_ at 26 and 31^{o}C.

Date inoculated	26^{o}C		31^{o}C	
	No. trees	Pct wilted	No. trees	Pct wilted
May 1977	6	100	6	0
June 1977	3	100	3	0
August 1977	5	100	5	0
June 1978	3	100	3	0
February 1979	3	100	3	0
May 1979	7	100	0	-
July 1979	8	100	0	-

Table 7.--Mean monthly temperatures in Kerrville, Texas during 1941-1970.

Month	Mean temperature over 29 years
January	7.5 C
February	9.6
March	13.0
April	18.3
May	21.9
June	25.6
July	27
August	27
September	23.9
October	18.6
November	12.5
December	8.8

Spring was very cool through June. Oak wilt developed rapidly in our established research plots. New infection centers also developed and spread rapidly. One new center was studied at the USDA Livestock Laboratory in Kerrville. The trees in this center had no history of wilt and all were full-crowned and apparently healthy in November 1978. The same trees were observed in June 1979 and found to be wilting very rapidly. Some had already been killed (Fig. 8). Fifteen trees were wilted and 3 were infected with C. fagacearum but not yet wilting. Eighteen trees in the center, including 2 of the 3 that were symptomless in June, had been killed by oak wilt in August 1979 (Fig. 9). One fungicide-treated tree survived in the center.

Other similar infection centers were observed in Bandera and Camp Verde. The rapid development of symptoms and death followed the same pattern in each location. The long cool spring season appeared to intensify oak wilt in Central Texas.

Survival of C. fagacearum over the hot summer in Central Texas was studied. One theory was that interior tree temperatures are cool enough for the fungus to survive.

Temperatures inside of diseased and healthy trees were measured in 1978. Small holes were drilled in root collars and in boles at d.b.h. (1.2 m above root collar). Holes were made on the four cardinal sides of each tree at each level. An electronic thermometer probe was inserted into each hole at various times and the temperature recorded. Ambient temperatures in the shade of test trees were also recorded. Comparisons of temperatures in 2 healthy and 2 diseased trees were made with ambient temperatures (Table 8). Interior tree temperatures were more stable than ambient temperatures.

Figure 8.--Group of live oaks with initial and rapid wilt development in spring 1979. Ceratocystis fagacearum was isolated from 2 living trees with symptoms in center and symptomless on each end of the group.

Figure 9.--All 18 trees shown in Figure 8 were dead in August 1979. The one surviving tree (last trunk on right) was treated with a fungicide in June.

Table 8.--Root collar and d.b.h. (1.2 m above root collar) temperatures inside healthy and diseased live oaks compared with ambient temperatures in Kerrville, Texas.

Date: Time of day	Root collar temp. Healthy	Root collar temp. Diseased	DBH temp. Healthy	DBH temp. Diseased	Ambient Temp.
7/18/79					
0800-0900	24.7	26.1	27.8	28.0	25
1530-1630	26.5	28.9	29.7	31.8	37.8
1800-1900	26.5	29.1	30.2	33.9	36.7
7/19/79					
0830-0930	24.8	25.9	27.5	27.6	26.7
1600-1700	26.2	28.7	29.7	31.9	36.1
1930-2030	25.9	29.1	30.8	34.4	35.0
Averages	25.7	28	29.3	31.3	32.9

Temperatures in healthy trees were lower and more stable than those in diseased trees at both levels. Root collar temperatures were consistently lower than d.b.h. temperatures. Temperatures in the lower bole and roots of healthy trees and in the roots of diseased trees were cool enough during two very hot summer days to support growth of *C. fagacearum*

Survival of *C. fagacearum* over the summer was compared in roots and twigs of previously wilted trees. Samples were taken from root flares and twigs of affected trees on July 18, 1978. The temperature exceeded 100^{0} F on that day. *Ceratocystis fagacearum* was isolated from the roots of 8 of the 23 sampled trees and from 2 of the twig samples. It survived the summer in infected trees but more frequently in roots than in twigs.

Survival of *C. fagacearum* and oak wilt development were greatly influenced by temperature. Oak wilt and fungal survival over the summer were more pronounced in 1979 than in the previous 2 years. Relatively low temperatures favored rapid wilt development, extension of the wilt season, and survival of *C. fagacearum* during July and August.

Spread and Distribution of Oak Wilt in Texas

Spread of oak wilt was observed in 32 research plots. Root-graft spread was confirmed by isolated *C. fagacearum* from roots connecting healthy and diseased trees. Insect spread was suspected but not confirmed. Root-graft spread was restricted to established infection centers and other means of spread were required for initiating new infection centers.

An infection center was studied after it was initiated in September 1977. It started with 12 trees that were 100% defoliated in November 1977 (Fig. 10). They all leafed out during the following spring but the crowns died shortly thereafter (Fig. 11). The number of trees in the center increased dramatically to include 32 trees in April 1978; 98 in June 1979; and 149 in November 1979. The infection center covered about 1.5 ha, 3.7 acres, after 2 years. There was extensive root grafting within this center and it appeared to be an important means of fungal spread. Evidence of other possible means of fungul spread was also observed. The two-lined chestnut borer (Agrilus bilineatus) was found in association with live oaks just before and after oak wilt symptoms developed in the center. Ceratocystis fagacearum was isolated from some of the galleries made by the insect. It was not isolated from living borers because none were collected for assay. The role of the two-lined chestnut borer in spreading oak wilt is still undetermined. There are effective means of spreading the fungus. Root-grafting is only one of them and others remain to be discovered.

An aerial survey of oak wilt between Kerrville and Bandera, Texas, was made in May 1978. Two flight lines were made and the larger areas with dead and dying live oaks were sketched on U. S. Geological Survey Maps. Sixty-eight major infection centers were sketched and they covered an estimated 850 ha (2,100 acres). The average size of each area was about 12.5 ha (31 acres). The largest area was about 152 ha (375 acres). In addition to the major infection centers, numerous small centers and individual trees with oak wilt were seen.

Figure 10.--Live oaks defoliated by wilt (trees on left) in a new infection center during November 1977. Living trees on right and in background are bordering the center. The same trees are shown in Figure 11.

Figure 11.--Expansion of the fall 1977 initiated infection center (seen in Fig. 10) during the following spring. The trees on left leafed out but died except for boles with sprouts. Trees on right and many in background became infected and died.

Tens of thousands of wilting and wilt-killed live oaks worth estimated millions of dollars to home owners, ranchers, and land developers were observed from the air. Developed areas, such as one near the River Club Estates in Kerrville, Texas, were left almost barren of desirable trees after wilt affected them (Fig. 12). Aerial surveys revealed large areas of live oaks killed by oak wilt.

A ground survey for oak wilt was made in April 1979. Wilt symptoms were difficult to see because most of the oaks observed had been defoliated by the spring canker worm. Ceratocystis fagacearum was isolated from live oaks in Austin, Bandera, Camp Verde, La Grange, and Waco. It was isolated from Spanish red oak in Midland by Dr. H. Kaufman (Texas Extension Service). Oak wilt was widespread in Texas.

Oak wilt was spread by root grafts and other undetermined means. It had killed hundreds of thousands of live oaks and was widely distributed across much of Texas.

Twig Blight and Dieback Caused by Botryodiplodia theobromae

Diseases other than wilt were also observed in Texas live oaks. Leaf spots, powdery mildew, and sometimes anthracnose were observed but did not cause any serious problems. Mistletoe was sometimes observed and heart rots were very common. Cankers, twig blights and dieback were the most conspicuous of the secondary diseases.

Small cankers and twig blight were observed in otherwise healthy live oaks at the LBJ Ranch and in Kerrville, Texas. Only small twigs were affected and they were scattered through crowns. Hail and insect wounds were commonly found on affected twigs. Botryodiplodia theobromae was consistently isolated from cankered tissues during 1976-1979. The cankers alone did not cause any serious damage to trees.

Figure 12.--Aerial photograph showing patterns of wilt spread in a high-value real estate zone near Kerrville, Texas. Most of the live oaks are dead (gray crowns). Light green crowns (lower right bldg.) are Spanish red oaks and shrubby dark green trees (bottom of photograph) are red cedar.

Healthy live oaks were inoculated with B. theobromae to see if cankers would develop. Pot-planted trees were wounded and inoculated with fungul mycelium in PDA. Sixteen were inoculated at 30^{o} C in 1977 and 1978. Fourteen developed cankers after 3 to 13 days. Cankers did not elongate very much but they girdled and killed trees at inoculation points. Botryodiplodia theobromae was pathogenic in healthy live oaks and produced cankers similar to those observed in the field.

Dieback was studied in trees that had suffered oak wilt. Trees that survived initial wilt infections were sometimes slowly killed by a progressive dieback and twig blight (Fig. 13). Botryodiplodia theobromae was isolated from 63% of 148 trees with dieback in 1976-1978. Trees partially killed and stressed by oak wilt appeared to be vulnerable to dieback from Botryodiplodia theobromae.

Botryodiplodia theobromae appeared to be a secondary pathogen that was sometimes involved in the final stage of "oak decline". It often finished killing trees that had already lost 50% or more of their crowns to oak wilt. The primary disease is oak wilt but B. theobromae also has a role in the etiology since it slowly killed trees already stressed by C. fagacearum.

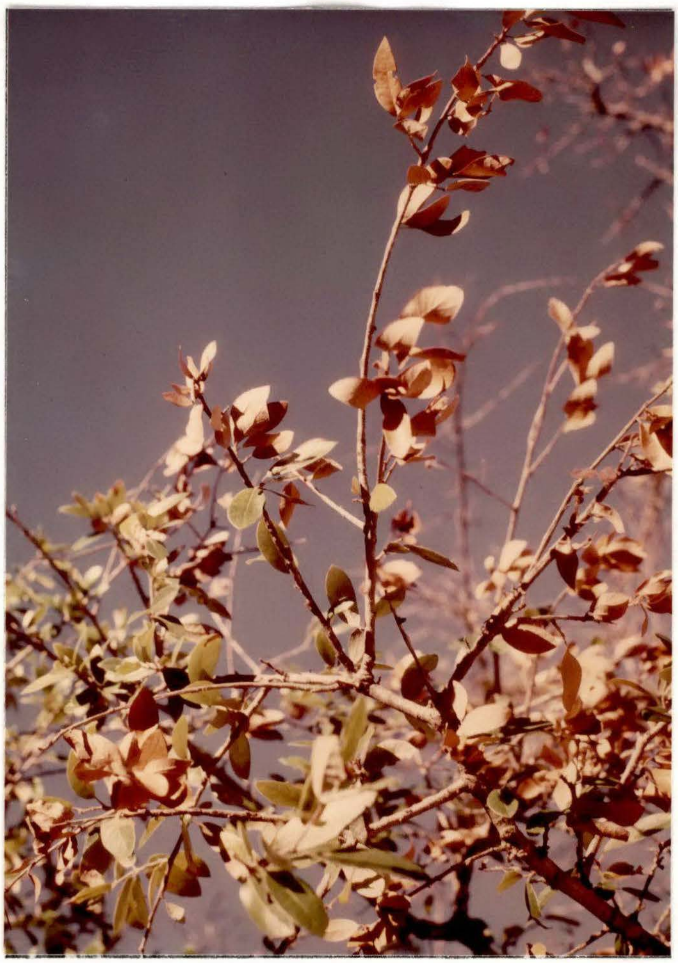

Figure 13.--Twigs apparently killed by Botryodiplodia theobromae in a live oak that previously had been weakened by oak wilt.

MANAGEMENT OF OAK WILT IN TEXAS

Selection of Fungicides For Possible Control

The effects of different fungicides on growth of *C. fagacearum* was tested in vitro. Fungicides were mixed in PDA at 1.0, 5.0, 10.0 and 25.0 μg (a.i.)/ml. Mycelial discs of C. fagacearum were placed on PDA with and without fungicides and the amount of growth after 10 days was measured. Arbotect™ 20-S (2-(4-thiazolyl) benzimidiazole hypophosphite) and Lignasan® (methyl-2-benzimidazole carbamate phosphate) completely inhibited growth of *C. fagacearum* at 1.0 μg/ml. Three other fungicides were not effective. Arbotect and Lignasan were highly effective against *C. fagacearum* in vitro.

Arbotect and Lignasan are water soluble. Arbotect is 20% active ingredient (a.i.) and Lignasan is 0.7% a.i. Both have been used and are labelled for control of Dutch elm disease. They were selected as potential weapons against oak wilt because of their effectiveness against the fungus in vitro and their established record of approved uses in American elms.

Prevention of Oak Wilt With Arbotect and Lignasan in Artificially Inoculated Live Oaks

Live oaks inoculated with *C. fagacearum* were treated with Arbotect and Lignasan to see if wilt could be inhibited. Nine pot-planted trees (about 1 cm in diameter) were inoculated with fungal spores and placed in a growth chamber at 26^{o} C. After one day, Arbotect was injected into 3, Lignasan into 3, and 3 controls received no treatment.

The 3 controls wilted in 15-20 days but symptoms did not develop in the fungicide treated. Both Arbotect and Lignasan inhibited symptoms in inoculated trees during the normal incubation period for the fungus.

The treated live oaks were allowed to remain in the 26^{o} C growth chamber for 60 days. Three leaves wilted within this period on one Lignasan-treated tree. Symptoms did not develop on the remaining trees.

Inoculated and treated trees were assayed for C. fagacearum after 60 days. The fungus was recovered from the root collar and roots of all treated trees though they did not have symptoms. Arbotect and Lignasan effectively prevented oak wilt symptoms from developing but did not kill the fungus throughout the tree.

Preliminary Applications of Lignasan in the Field

Live oaks with incipient and advanced but inactive wilt symptoms were injected with Lignasan in May 1977. Two live oaks infected with C. fagacearum at the LBJ Ranch were injected with 65 ml Lignasan/cm d.b.h. The Lignasan was diluted with 4 parts water. The solution was injected rapidly with high pressure (65 psi). Additional symptoms did not develop in the trees and C. fagacearum could not be recovered after 1 year. Eight trees with advanced but inactive wilt at Kerrville, Texas, were difficult to inject. Some vessels had been made non-functional by wilt and only a small amount of liquid could be injected in a 24-hr period. Therefore, concentrated Lignasan was used rather than a dilution. The amounts injected varied from 6-20 ml/cm d.b.h. Some of the trees responded to the treatment with larger and greener leaves and others showed little or no change. All treated trees were still living in 1979 but 6 of the untreated controls were dead (Fig. 14). Lignasan apparently stopped wilt from developing in 2 trees

Figure 14.--Live oaks treated with Lignasan in May 1977 and still surviving in June 1979 (left side of road) compared with untreated controls (dead trees on right side of road).

infected with *C. fagacearum* and prevented 8 trees with advanced but inactive wilt from dying after 2-1/2 years.

Summer applications of Lignasan were made in July 1977. Four live oaks that had initial wilt in May 1977 were selected. Two were injected with Lignasan (65 ml/cm) diluted in 1 part water and 2 were used as untreated controls. The treated did not develop additional symptoms after 2 years but the two controls died during fall 1977. Twelve trees with advanced "decline" were also injected in July. The history of wilt in these trees was not known. Treatments were difficult due to clogged vessels but an undetermined amount of undiluted Lignasan was injected into each tree. There was a little improvement in the appearance of these trees after 2 years. Dieback was stopped and the new leaves were greener and larger than in untreated controls. Seven controls were still alive but 5 were dead after 2 years. Lignasan appeared to be effective when applied during summer.

Six live oaks with incipient wilt were treated in November 1977 and compared with 6 untreated controls. Each tree was infected with *C. fagacearum*. The amount of wilt was about the same in both treated and controls in April 1978. New symptoms also developed and *C. fagacearum* was isolated from both treated and control during the spring. Fall application of Lignasan in wilting live oaks was not effective in 1977.

Preliminary applications of Lignasan in the field indicated that oak wilt could be stopped or prevented under certain conditions. Summer and late spring applications gave the gest results and late fall applications appeared to be ineffective.

Application of Arbotect and Lignasan in Trees With Different Degrees of Oak Wilt

Experimental treatments were refined as more was learned about the etiology of live oak wilt. Experiments were designed to see if the degree of wilt could affect treatment success.

Trees with incipient wilt in November 1977 were treated in May 1978. The trees were 100% defoliated in November and *C. fagacearum* was isolated from them. They all leafed out with very small chlorotic leaves, thin crowns and very little dieback in April. Four of the trees were injected (high pressure) with Lignasan at 65 ml/cm d.b.h. in one part water; 4 were injected with Arbotect at 6.5 ml/cm d.b.h. in 4 parts water; and 4 controls received no treatments. Uptake was slow in each tree. Two Arbotect treated and the 4 controls died during spring and summer and the remaining treated trees were 50-75% dead in August 1978. The boles of 4 Lignasan and 2 Arbotect treated trees were still alive in November 1979. A symptomless live oak adjacent to 2 Arbotect treated trees failed to develop wilt after 2 years but the surrounding trees wilted and died (Fig. 15). The tree might have been protected by Arbotect injections into adjacent trees which were root-grafted to it. Infected trees with advanced wilt were not helped by fungicide treatments in spring 1978 but there were indications that fungicides might prevent wilt from developing in symptomless trees adjacent to infected ones.

Figure 15.--Healthy live oak surrounded by oak-wilt-killed trees. The tree might have been protected by fungicides received through root grafts with adjacent Arbotect-treated trees on left and right.

Twelve symptomless live oaks on the perimeter of an infection center were pre-selected in May for August 1978 treatments. Four were marked for Arbotect; 4 for Lignasan; and 4 for untreated controls. Treatments were made in August as planned. Uptake was very slow in the 4 Arbotect-treated but was fast in the Lignasan-treated. Treatments were evaluated in spring and fall 1979. The 4 Arbotect-treated and controls failed to leaf-out in the spring and were dead to the root collar in August but the 4 Lignasan-treated did not develop wilt and were still healthy in November. Trees surrounding the Lignasan treated developed wilt and died in 1979. Lignasan prevented wilt from developing in relatively healthy uninfected trees but Arbotect had no beneficial effects in trees that were apparently already stressed by clogged vessels on the perimeter of an active infection center.

A new low-pressure injection procedure was used in 1979. An injector developed by the Elm Research Institute was used to inject fungicides into root flares at 30 psi.[1/] Injection points were made at 6-inch (15.24 cm) intervals around the circumference of the tree. Lignasan was injected at the rate of 74 ml/cm d.b.h. (about 1 qt per each 5 in d.b.h.) and Arbotect at 7.4 ml/cm d.b.h. (about 3.2 ozs per each 5 in d.b.h.). This method and these rates were used in all 1979 treatments.

1/ The Conscientious Injectors Handbook - Elm Res. Inst., Harrisville, NH. 03450.

Live oaks with varying degrees of wilt were treated in June 1979. Trees with 5 classes of symptoms were used. Symptom classes were: (a) symptomless but infected with C. fagacearum; (b) initial wilt symptoms; (c) early stage of wilt with most of crown affected; (d) moderate wilting over entire crown, and (e) severe wilting with all leaves showing symptoms. All of these trees were affected by wilt for the first time in spring 1979. One tree in each class was treated with Arbotect, one with Lignasan; and one untreated served as a control. Fungicide uptake varied among trees due to differences in the degree of wilting. Uptake varied from very fast in symptomless to very slow in severely wilted trees.

Treatments were most effective in symptomless infected trees and in trees with initial symptoms but they were also beneficial in trees with early and moderate stages of wilt (Table 9). Figures 8 and 9 show symptomless live oaks infected with C. fagacearum and later killed by wilt. Only one live oak survival in the group and it was treated with Arbotect (Fig. 9). Figures 16, 17, and 18 illustrate the effectiveness of fungicide treatments. The fungicides were not effective in trees with severe wilt (Figs. 19 and 20). Both Arbotect and Lignasan stopped oak wilt development except in severely wilted trees.

Figure 16.--Symptomless live oak (center) infected with Ceratocystis fagacearum and tree with moderate wilt (branches at upper right corner) treated with Lignasan in June 1979. Tree on left had severe wilt symptoms and served as an untreated control. Compare with Figure 17.

Figure 17.--Comparative photograph made in August to show responses of live oaks to June 1979 treatments (Fig. 16). Dead tree on left (white ribbon on trunk) was a control with severe wilt; healthy tree on right (red ribbon) was treated with Lignasan and was symptomless in June; tree branches (upper right) showed moderate wilt when treated in June but wilt had stopped in August.

Figure 18.--This live oak had moderate wilt symptoms when treated with Arbotect in June but the wilt was stopped and leaves greener when the photograph was made in August 1979. The crown was thin but alive and only small twig dieback developed.

Figure 19.--Live oak with severe wilt (center and with red ribbon) and early stage of wilt (larger trunk on left) treated with Lignasan in June 1979. All leaves on center tree were wilting and some were turning brown. Compare with Fig. 20.

Figure 20.--Comparative photograph made in August to show response of live oaks to June 1979 treatments (Fig. 19). Dead tree on right was severely wilted and tree on left was in early stage of wilt when treated with Lignasan. Wilt was stopped in tree with early stages.

Table 9.-- Treatment of Kerrville, Texas live oaks with Arbotect (7.4 ml/cm d.b.h.) and Lignasan (74 ml/cm d.b.h.) for control of oak wilt in June 1979.

Tree condition when treated	Treatment	Tree condition in Nov. 1979
Infected with <u>Ceratocystis fagacearum</u> but no symptoms (Very fast uptake of fungicides)	Arbotect	Wilt inactive; 1 dead limb
	Lignasan	No symptoms
	Control	Dead
Initial symptoms - less than 10% defoliated (Very fast uptake)	Arbotect	Wilt stopped - no dieback
	Lignasan	Wilt stopped - no dieback
	Control	Wilt active; 50% crown dieback
Early stage of wilt Most of crown affected (Fast uptake)	Arbotect	Wilt stopped; large leaves;no dieback
	Lignasan	Wilt stopped; no dieback
	Control	Dead
Moderate wilting over entire crown (Slow uptake)	Arbotect	Wilt stopped; thin crown;little dieback
	Lignasan	Wilt stopped; thin crown;little dieback
	Control	Dead
Severe wilting All leaves showing symptoms (Very slow uptake)	Arbotect	Wilt active; 50% crown dieback
	Lignasan	Dead
	Control	Dead

Preventative treatments were applied to symptomless live oaks within 30 m (98 ft) of infected trees. The fungicide application rate was one-half the regular treatment. Seven trees were treated with Arbotect, 3 with Lignasan, and 2 served as controls in June 1979. Similar treatments were made in August when 3 trees were treated with Arbotect, 4 with Lignasan and 3 served as controls. Treatments were evaluated in November. Wilt did not develop in any Lignasan-treated, 6 of the 10 Arbotect treated, and 1 of the 5 controls (Table 10). One Arbotect-treated tree was killed and 3 developed wilt in a few limbs only. Most controls were severely wilted or dead. Wilt was prevented or reduced by fungicidal treatments in trees that were expected to develop it.

Six infection centers and 146 live oaks were treated with Lignasan in Bandera, Texas during August 1979. The response to treatment was positive when observed in November. Rapidly advancing infection centers were apparently stopped (Fig. 21). New growth on some infected trees was healthy but trees with severe wilt did not respond well. The treatments will be evaluated more closely in 1980.

Fungicides were effective when used to prevent or stop oak wilt development. The rate and time of application and degree of wilting all affected treatments. Summer applications were best but late spring applications were also desirable. Trees with initial wilt symptoms responded well but trees with severe wilt were difficult to inject and did not respond well.

Figure 21.--Live oaks treated with Lignasan for control of oak wilt in August 1979. The infection center was apparently stopped by the treatment since it had not advanced when observed in November. The dead and cut trees in background were killed by oak wilt in the spring. Tree with lighter green color was also infected but still survives.

Table 10.--Treatment of symptomless live oaks near infected trees with Arbotect and Lignasan for prevention of oak wilt in 1979.

Treatment	No. trees	Condition in November
June		
Arbotect	7	4 - no wilt; 2 - wilt in few twigs; 1 - dead
Lignasan	3	No wilt
Control	2	1 - 75% crown dieback; 1 - dead
August		
Arbotect	3	2 - no wilt; 1 - wilt in one limb
Lignasan	4	No wilt
Control	3	1 - no wilt; 1 - severe wilt; 1 - dead

Discussion of Alternatives to Fungicide Treatments and a New Approach For Managing Oak Wilt

Oak wilt spread through root grafts has been slowed by destroying or killing root connections between healthy and infected trees. This method has been used in commercial forests in northerly states. It might also work in Texas but will affect only root transmission. Killing infected trees with commercial silvicides might also slow the spread of oak wilt by killing roots and indirectly reducing the wilt fungus population. Adjacent healthy trees might be killed by this treatment since silvicides can be translocated to them through root-grafts. Application of these treatments might be desirable in large wilt affected areas where fungicide treatments would be impractical.

Elimination of root-graft spread will slow but not stop the advancement of oak wilt. Insects, man, and other biological or physical forces can spread the fungus also, but destroying root-grafts will not affect them. Management rather than elimination of insects and the activities of man can reduce oak wilt. Insect vectors have not been identified but evidence of their presence has been seen. The activities of man and their suspected impact on oak wilt have been seen also. Sap from pruned and wounded trees might attract insect vectors during spring. Wood piles made from trees killed by oak wilt might harbor insect vectors which emerge and start new infections in different locations. Transport of infected trees by man also helps spread oak wilt to new areas. These activities can be minimized to reduce the chances for oak wilt spread. Localized infections in tree crowns can be pruned out before the infection becomes systemic.

New concepts for oak wilt control should utilize the physical and biological forces of nature present on site. High temperatures are lethal to *C. fagacearum*. These temperatures apparently build up in parts of tree crowns but not in lower trunks during summer in Texas. Collection and transfer of heat from the sun to tree trunks and root collars might increase interior temperatures enough to kill the fungus without adversely affecting trees. This theory has not been tested. Another concept is that high temperature attenuates *C. fagacearum* and might make it easier to kill with a smaller amount of fungicide. If this concept is true, it would be useful for an integrated pest management program. Fungicides were least effective when applied during cool spring and fall periods and most effective when applied during warm to hot periods. Oak wilt control programs should take advantage of high summer temperatures.

High temperatures have a negative effect on *C. fagacearum* but promote growth of some other fungi. Other fungi might play an important role in reducing the incidence of oak wilt. They might serve as a means of natural biological control.

Healthy live oaks were occasionally observed adjacent to trees killed by oak wilt. They were connected by root-grafts but were not infected (Fig. 22). These trees might have escaped oak wilt infection, been resistant to it, or some other unexplained factor might have prevented wilt.

Fungi were isolated and identified from healthy live oaks in oak wilt centers. Some of the interesting ones were *Cephalosporium* sp., *Penicillium* sp., *Trichoderma* sp., and *Verticillium* sp. Pot-planted live oaks were inoculated with *C. fagacearum* spores and then spores of

Figure 22.--Healthy live oaks 2 years after surrounding trees were killed by oak wilt in Kerrville, Texas.

the above-listed fungi. The trees were placed in a 26° C growth chamber with 12 h. day-night simulations. All trees developed wilt symptoms within 20 days but the one inoculated with Cephalosporium stopped wilting when the top one-third had been killed. Additional wilt did not develop in the tree after 60 days but the remaining trees in the study were killed to the roots after 25 days. Three symptomless live oaks that were inoculated with Cephalosporium diospyri in February 1979 were inoculated with C. fagacearum spores in July. Two did not develop oak wilt after 60 days and one slowly developed wilt symptoms in about 50% of its leaves. Cephalosporium diospyri was the only tested fungus that interfered with oak wilt development in trees inoculated with viable C. fagacearum spores. Data collected thus far are insufficient to make statements about using Cephalosporium spp. for suppression of oak wilt but future research should include it along with other fungi as possible biological agents for use in an integrated oak wilt management program.

Discussion and Conclusions

Live oaks have died rapidly in Texas for 45 or more years. Early research reports described symptoms and the rate of disease development but failed to identify the causes. More recent reports identified Cephalosporium diospyri as the primary cause and named the disease "live oak decline". The "decline" was reportedly a highly evolved vascular wilt that required 10 or more years to kill trees (Van Arsdel et al. 1974). This report conflicted earlier descriptions of the disease which killed live oaks within weeks (Taubenhaus 1934; Dunlap and Harrison 1949; Halliwell 1964). Our research in 1977-79 was to resolve some of the questions about "live oak decline".

Cephalosporium diospyri did not cause wilting or "decline" in healthy inoculated live oaks but it colonized them. Live oaks in Florida, Louisiana, Mississippi, and Texas were colonized by C. diospyri and other non-pathogenic fungi but did not wilt. Kaufman (1978) also reported healthy live oaks colonized by C. diospyri.

Cephalosporium diospyri is best known for causing persimmon wilt. Its distribution was known in Alabama, Florida, Georgia, Mississippi, South Carolina, Tennessee, and Texas as early as 1939 (Toole and Lightle 1960). "Oak decline" was not and still has not been reported in many of these areas where C. diospyri was found. Dunlap reported C. diospyri in Texas persimmon (1939) but did not associate it with the live oak disease that he studied for the following 10 years (Dunlap and Harrison 1949). This fungus is a virulent pathogen in persimmon but is not the cause of live oak wilt or "decline".

Oak wilt, caused by Ceratocystis fagacearum, is the most important lethal disease of oaks in North America. It was first identified in Wisconsin (Henry and Moses 1943) and later found in other northern states. Its distribution in the South was believed to be limited or restricted by high summer temperatures (Bretz and Morison 1953; Rexrode and Lincoln 1965; Tainter et al. 1974). Temperatures of 32^{o} C (89.6^{o} F) and above are inhibitory or lethal to C. fagacearum (Houston et al. 1965) but Dooling (1961) found it in Dallas, Texas, where summer temperatures sometimes exceed 38^{o} C (100^{o} F). Van Arsdel et al. (1975) disputed the report of oak wilt in Dallas. They reported that either the original identification was an error or the fungus had been killed by high temperatures and concluded that Ceratocystis fagacearum does not survive in Texas.

Ceratocystis fagacearum was isolated from live oaks exhibiting essentially the same symptoms described by Taubenhaus, Dunlap and Harrison, and Halliwell. Disease outbreaks were in spring and fall as reported by Dunlap and Harrison (1949). I believe that oak wilt might have been observed but not identified by Taubenhaus in 1934 when he first reported the live oak disease at Austin. However, oak wilt and its cause had not been identified anywhere in the world at that time. It was finally identified in Wisconsin during the early 1940's.

Oak wilt spreads slowly and distances of 50 feet (15.2 m) or more are considered long (True et al. 1960). Infection centers at Kerrville, Texas expanded to affect many trees in a dense stand within 2 years but the overall distance covered was relatively short. There is almost a continuous 25 mile stretch of oak wilt from Bandera to Kerrville. Oak wilt spots also extend from Austin to Kerrville and many other areas. Considering the limits placed on oak wilt by high summer temperatures in Texas, the fungus has had a highly efficient means of spread. Its wide distribution must have taken many years and could have started even before 1934.

The total distribution of oak wilt in Texas is not known but it appears to be quite extensive. We have positively identified it in Austin, Bandera, Camp Verde, La Grange, Midland, and Waco. It was identified in Dallas (Dooling 1961) where it is now less obvious than in any of the other locations. I believe that Toole and Filer (1966) might have observed it and isolated the fungus during a 1-week survey in April 1966. They isolated *Chalaropsis* and suggested that Texas A&M University Cooperators investigate it and other fungi from wilting live

oaks in Angelton, Sinton, and Yoakum. They described and photographed symptoms like those we associated with oak wilt and found trees with active wilt during the cool spring. They found disease outbreaks in Angelton, Columbus, Hallettsville, Kerrville, Sinton, Temple, and Yoakum. Extensive surveys should reveal many other locations with oak wilt. The state needs to know the distribution of oak wilt if it plans to try suppressing it.

Oak wilt management in Texas should include fungicide applications, destruction of root-grafts, killing infected trees and use of biological agents to suppress wilt when found. Systemic fungicide applications might be most effective during warm and hot periods. Trees with advanced wilt are difficult to inject and treatments are ineffective in them. This is primarily due to clogged vessels which impair water movement. Dr. Tainter[2/] reported that translocation of non-structural carbohydrates from leaves and twigs into the sapstream is less in wilted than in non-wilted live oaks. This finding indicates a reduction in sap flow which might adversely affect movement of fungicides down into the roots. Root infections, which allow summer survival, are difficult to impossible to control with fungicides. Infected root-grafts between healthy and diseased trees should be destroyed.

2/ F. H. Tainter, Clemson Univ., Cooperative Study No. 19-270; Southern Forest Experiment Station and Univ. Arkansas. "The effect of tree vigor on growth and storage of non-structural carbohydrates as related to oak decline".

Silvicides that kill roots should slow oak wilt spread when applied to wilting trees. Once trees are wilted they are rapidly colonized by other fungi. Cephalosporium spp. and/or Dothiorella spp. (Ames and True 1967), Hypoxylon punctulatum and Phialophora spp. (Wood and Peterson 1959) have been associated with oak-wilt killed trees in other states. Some of these fungi might serve as a biological means of oak wilt suppression. Even Cephalosporium might help suppress oak wilt in Texas. Its effects on oak wilt are not understood but some Cephalosporium spp. have been recognized as having antibiotic properties (Waksman and Horning 1943). All available means of wilt suppression should be considered for an integrated management program.

LITERATURE CITED

1. Bretz, T. W. and D. W. Morison. 1953. Effect of time and temperature on isolation of the oak wilt fungus from infected twig samples. Plant Dis. Rep. 37:162-163.
2. Dooling, O. J. 1961. Oak wilt identified in Texas. Plant Dis. Rep. 45:749.
3. Dunlap, A. A. 1939. Cephalosporium wilt of persimmon in Texas. Plant Dis. Rep. 23:347.
4. Dunlap, A. A. and A. L. Harrison. 1949. Dying of live oaks in Texas. Phytopathology 39:715-717.
5. Halliwell, R. S. 1964. Live oak decline. Proc. 40th. Int. Shade Tree Conf. P. 178-180.
7. Halliwell, R. S. 1965. Oak decline in Texas. (Abstr.) Phytopathology 55:1060.
8. Halliwell, R. S. 1966. Association of Cephalosporium with oak decline in Texas. Plant Dis. Rep. 50:75-78.
9. Henry, B. W. and C. S. Moses. 1943. An undescribed disease causing rapid dying of oaks. (Abstr.) Phytopathology 33:18.
10. Houston, D. R., C. R. Drake, and J. E. Kuntz. 1965. Effects of environment on oak wilt development. Phytopathology 55:1114-1121.
11. Kaufman, H. W. 1978. A comparison of reactions to <u>Cephalosporium diospyri</u> in four tree species. Ph.D. Dissertation. Texas A&M Univ., College Station, Texas, 73 p.
12. Rexrode, C. O. and A. C. Lincoln. 1965. Distribution of oak wilt. Plant Dis. Rep. 49:1007-1010.
13. Tainter, F. H., M. Tucker, B. Washburn and J. Tiner. 1974. Oak wilt remains static in Arkansas. Plant Dis. Rep. 58:622-624.

14. Toole, E. R. and T. H. Filer, Jr. 1966. Oak decline in Texas--April 1966. U. S. For. Serv., South. For. Exp. Stn., Hardwood Insect and Dis. Proj. (RWU-2209, Stoneville, Miss.). Proj. Rep. (Proj. file) 9 p.

15. Toole, E. R. and R. C. Lightle. 1960. Status of persimmon wilt, 1959. Plant Dis. Rep. 44:45.

16. True, R. P., H. L. Barnett, C. K. Dorsey, and J. G. Leach. 1960. Oak wilt in West Virginia. West Virginia Agric. Exp. Stn. Bull. No. 448T, 119 p.

17. Van Arsdel, E. P. 1970. Live oak decline, its identification and possibilities of control. Proc. 3rd. Ann. Texas Conf. on Insect, Plant Dis., Weed and Brush Control. P. 56-57.

18. Van Arsdel, E. P., D. L. Bush, and T. W. Jares. 1974. Previsual detection of oak decline and rust diseases in Texas with infrared photography. Proc. Am. Phytopath. Soc. 1:110.

19. Van Arsdel, E. P., D. L. Bush, and H. W. Kaufman. 1975. Comparison of Cephalosporium diospyri from Texas oaks with Ceratocystis fagacearum. Proc. Am. Phytopath. Soc. 2:142.

20. Waksman, S. A. and E. S. Horning. 1943. Distribution of antagonistic fungi in nature and their antibiotic action. Mycologia 35:47-65.

21. Wood, F. A. and J. E. Peterson. 1959. Fungi isolated from oak-wilt-infected and apparently healthy oak trees. (Abstr.) Phytopathology 49:555.